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Harvard's Indoor Air Pollution/Health Study

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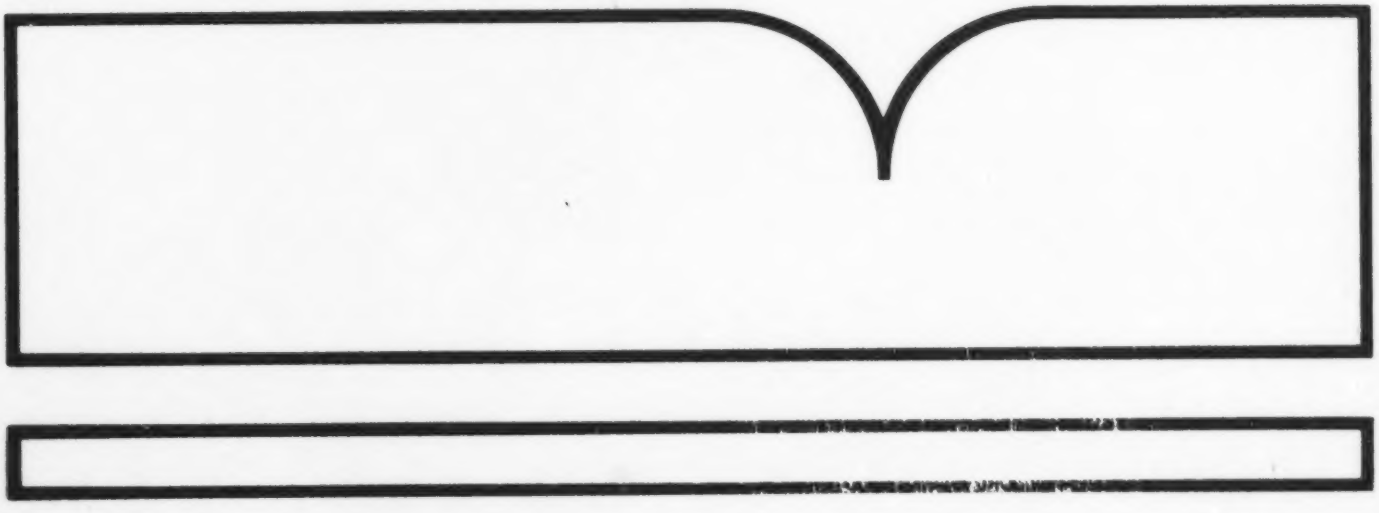

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by

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16. ABSTRACT

An indoor air pollution/acute respiratory health study is being conducted by researchers at the Harvard University School of Public Health. Upper and lower respiratory symptoms of 300 children living in Watertown, Massachusetts, have been recorded on a daily diary by a parent. Every two weeks the parent is called for the illness/wellness history. At the end of the month the calendar is returned by mail. Families participated in this survey from the time of induction (after September 1, 1984) to August 31, 1985. Coincident with the symptom survey, indoor air pollution measurements are made in each home and in the elementary schools of the participating student. Participants were selected by stratified random sampling. The original pool of possible participants was all children in the 3rd and 4th grades from Watertown who had been tested for lung function during the previous year. The questionnaire describing home characteristics provided information on type of cooking stove and number of smokers in the home. Sampling was stratified by presence or absence of a gas stove and by presence or absence of a smoker. Because previous work clearly shows unequal variance in indoor NO_2 and particle concentrations within these strata the gas stove and smoker strata were oversampled. This paper reports the preliminary findings from the Watertown indoor survey. It describes the design modifications implemented in the two cities currently being studied; Kingston, Tennessee and St. Louis, Missouri.

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INTRODUCTION

Indoor airborne contamination may be several times higher than outdoor concentrations and even exceed National Ambient Air Quality Standards.[1] However, health effects associated with indoor exposures to air pollutants emitted from indoor unvented combustion sources are not well documented. One exception may be the effects of carbon monoxide (CO). The Harvard Air Pollution Health Study is engaged in a cross-sectional, as well as longitudinal, investigation of respiratory disease and pulmonary performance changes in a population of approximately 10,000 adults and a similar number of children.[2] A component of this study is the investigation of the respiratory effects of indoor (home) combustion-related air pollution. The current work focuses on NO_2 and respirable size particulates (PM 2.5) less than 2.5 um in diameter emitted primarily from unvented gas and kerosene appliances and tobacco smoke. Between the winter of 1984 and summer of 1988 approximately 1800 children will be surveyed for daily respiratory symptoms over the course of a 9-12 month period. Their homes, schools and the outdoor environment will be monitored repeatedly over the same time period.

Increased reporting of respiratory symptoms as well as deficits in pulmonary function in children whose parents smoke have been reported previously.[3,4,5] The literature is not as consistent with regard to respiratory health effects associated with gas cooking stoves and kerosene burners. Vedal[6] reviewed the gas stove studies and indicated that with the exception of three studies, all others did not have enough subjects to identify a significant effect of less than a 50% increase in risk.

The basic limitation of the epidemiologic investigations of indoor pollutants is the reliance on questionnaires and categorical variables to define exposures. Within a community, indoor and personal exposure studies of NO_2 and respirable particles (RSP) have revealed poor correlations of those measures with concurrent outdoor measurements.[7,8,9,10] Using home indoor concentrations of NO_2 and RSP to predict integrated personal exposures resulted in significant squared correlation coefficients (R^2) ranging between 0.6 and 0.8. Subgroups within a population had $R^2 > 0.8$ for home and personal measurements. Time weighted concentrations using indoor home and school and outdoor concentrations and a fixed effect for other microenvironments showed some marginal improvement in prediction.

These and other air pollution exposure studies indicate the potential for misclassification of subjects in an epidemiologic study when relying exclusively on descriptor variables. The issue of misclassification of disease and/or exposure and the resulting effect on the statistical analysis has been examined in several papers including Shy et al 1978, Copeland et al 1977, Gladen and Regan 1979 and Ozkaynak et al 1984.[11,12,13,14] Ozkaynak et al 1984 used data and models derived from our studies. The analysis indicated that reliance on categorical variables underestimates the true variance which is responsible for overlap in exposures among groups. The overall effect is to reduce the statistical power of studies which use questionnaires exclusively to determine air pollution health effects. The ratio of relative risk of disease across exposure classes may be in error by a factor of two to seven depending on the prevalence of disease, subjects studied and contaminants (particles vs. NO_2). Our current study is designed to overcome this limitation by directly measuring indoor air pollution (homes and schools) as an approximation to a child's exposure.

STUDY DESIGN

The sample was drawn from children currently entered in a new cohort of the Harvard Air Pollution Respiratory Health Study. Approximately 1000 children are to be identified in each city. From these households, 300 will be selected in each community for the diary monitoring study. For example, children in Watertown, Massachusetts in grades three and four were initially examined in the fall of 1983. Using information collected on questionnaires, four categories of characteristics were designated based on parental smoking and gas cooking. Table I indicates the category description and stratification of the new cohort at the initial selection of sample through to the actual sample participating in health and indoor air quality surveys. Approximately 10% of the houses were reclassified following the direct assessment by the field survey team visiting homes.

The study was divided into two sections; Acute Health Survey and Indoor Air Monitoring Sampling. The Acute Health Survey was designed to obtain a daily record for participating children of respiratory symptoms and their severity. Table II lists the symptoms which the child's parent recorded on the calendar. Each day a symptom was present, the code was recorded on the calendar. An interviewer called the home twice a month for a report of health calendar entries. At the end of every month each home received an envelope for mailing the calendar back to the study office.

The Air Monitoring component was designed to estimate a child's exposure by determining the air pollution concentration of NO_2 and respirable size particles in three microenvironments: indoor at home, outdoors at a central site and satellite sites and in the school classroom. Each home was monitored for two consecutive weeks during a winter season and again for two consecutive weeks during a summer season. The outdoor monitoring consisted of the continuous recording NO_2 and nephelometer instruments at our central site. In addition, two PM 2.5 particle monitoring sites were established to obtain consecutive week-long samples. NO_2 was measured outside each home simultaneously with the home sampling. Indoor measurements were obtained at six elementary schools. Twenty-six classrooms were monitored for NO_2 and six classrooms (one per school) were monitored for PM 2.5. On Monday morning, the cooperating teachers uncapped two samplers and placed them in an established location. Friday afternoon a field technician visited the school to retrieve the NO_2 and PM 2.5 samples. The technician left new NO_2 samplers, changed the particle filter and reset the sampler to collect a 40 hour sample (8 hour/day) during the following week. Schools were monitored from February to June except during vacation weeks in February and April.

Table I. Home Characteristics for the Harvard Indoor Air Quality Health Survey in Watertown, Massachusetts

Key	Group	No. in New Cohorts at Outset	Target for Indoor Health Survey	Actual No. in Health Survey at End of Intake	Winter Sample Completing Health Survey	Winter Sample as Coded by Indoor (IAQ) Survey*
No Gas No Smoke	1	82 (13%)	28 (09%)	26 (08%)	22 (09%)	29 (11%)
No Gas Smoke	2	112 (17%)	66.5 (21%)	61 (19%)	57 (21%)	49 (18%)
Gas No Smoke	3	210 (33%)	66.5 (21%)	54 (17%)	56 (21%)	55 (21%)
Gas Smoke	4	234 (37%)	155 (49%)	140 (44%)	132 (49%)	134 (50%)
		638	316	281	267	267

* 10% change in home classification code

Table II. The list of symptoms recorded by parents for each child participating in diary survey of respiratory illness.

A Hoarseness
B Sore Throat
C Cough
D Phlegm from the Chest
E Pain in the Chest
F Wheezing
G Fever
H Ear Pain or Discharge
J Runny or Stuffed Nose
K Burning, Aching or Red Eyes
L Restricted Activities
N Saw Doctor or Nurse
P Hospitalized
O Healthy or None of the Above

Study Schedule

Letters were sent to families starting October 1984. Twenty letters were sent each successive business day until the end of November. Families were then contacted by phone to explain the study and solicit participation. First attempts to contact the families were made starting five days after the letters were mailed. When a family agreed to participate, a health calendar was mailed to the participant and the family was then contacted by phone twice per month until August 31, 1985.

The target window for the winter indoor sampling was established as beginning the week of November 15, 1984 and ending the week of March 15, 1985. Due to delays in the manufacture of sampling equipment, initial monitoring did not begin until mid-December. Starting in the middle of December, 42 homes were set-up over a two week period to act as a pilot and training activity. Each home was sampled for three consecutive one-week periods. Full-scale winter sampling began in January 1985. Attempts were made to maintain the targeted stratification of the four categories during each sample week. The final completion of sampling in the winter season was at the end of April. The summer sampling session commenced in mid-May and ended in late September.

Sampling Protocol

Approximately 20 homes were set-up each week during the sampling period. Sample kits were assembled at Harvard containing prenumbered and assigned samplers with all forms necessary for the sample week. The sample kit was then assigned to the home at the set-up visit. Each home was sampled for nitrogen dioxide, water vapor, ventilation and particle concentrations. A detailed questionnaire was administered and a complete floorplan with interior dimensions was obtained by the field technician.

Nitrogen dioxide was measured using four passive diffusion tubes.[15] These monitors were uncapped and placed in the kitchen, activity room, child's bedroom and outside the home. Samples were prelabelled and assigned by a computer program. This program randomly assigned 5% replicates and 5% field blanks to the field sampling kits. A passive diffusion water vapor tube was placed with the NO_2 monitor in the activity room. Also placed in the activity room was a Harvard Aerosol Impactor (PM 2.5).[16] A timer was set to run the PM 2.5 sampler for the entire one-week period except during the eight hours each weekday the child was expected to be in school. Air exchange measurements were obtained using a perfluorocarbon tracer (PFT) system.[17] To achieve more uniform distribution of tracer gas, two small permeation caps containing a single type of PFT were placed two per floor. Passive charcoal receptor tubes were then located to measure the concentration of the emitted gas.

One week after the set-up visit the technician returned to the home to change samplers for the next monitoring week. One week after the change visit the samplers were collected and the set up was removed by the technician. During our normal sampling schedule, approximately 20 homes were set-up for monitoring, 20 homes visited to change samples and 20 homes visited to pick-up monitoring equipment during each week.

DESCRIPTIVE RESULTS

This paper reports only the results of the winter sampling in Watertown, Massachusetts. The purpose is to document protocol and illustrate analyses that will be performed on the emerging more comprehensive data.

It was intended that homes of all the participants in the Acute Health Survey would be monitored. However, because of dropouts, refusals or difficulties scheduling appointments, only 267 (84%) of the homes were set-up during the winter cycle. Table III presents the winter sample of participating homes by stratification variables.

Table III. Number of homes with at least one valid NO_2 and PM 2.5 measurement per week for Watertown winter sampling.

	WEEK 1	WEEK 2
GAS COOKING	150	157
ELECTRIC COOKING	67	69
PARENTAL SMOKING	147	152
NO PARENTAL SMOKING	70	74

The distribution of NO_2 concentrations within and outside homes was similar between weeks one and two of sampling. Paired t-tests indicated no differences in the means between weeks. Therefore, only Week 1 results for gas cooking homes and non-gas cooking homes are presented in Tables IV and V. For gas cooking homes the concentrations across the distribution were higher in the kitchen than in the activity room and bedrooms. The mean, median and upper percentiles were all higher indoors than outdoors. Only in the lower percentiles was the outdoor concentration higher then the indoor bedroom concentrations. For non-gas cooking homes, the concentrations were systematically lower indoors than outdoors. The bedroom concentrations appeared to be slightly less than both the activity room and kitchen concentrations across the distribution. In comparing distributions of NO_2 concentrations between gas and non-gas cooking homes, there was an overlap. The lower 10 percent of gas cooking homes had NO_2 concentrations that overlapped the upper 25 percent of the non-gas cooking homes. The outdoor concentrations of NO_2 in Watertown were spatially uniform.

Table IV. NO_2 concentrations (ppb) for Watertown, MA in gas cooking homes during the first week of winter sampling period.

				Percentiles					
	N	MEAN	SD	10	25	50	75	90	MAX
BEDROOM	189	25.2	11.7	12.9	17.5	23.2	30.3	39.6	98.1
ACT. ROOM	187	28.2	13.2	14.6	19.0	26.9	32.7	43.6	108.7
KITCHEN	188	40.6	18.4	20.3	27.5	37.5	51.5	64.8	132.8
OUTDOOR	180	21.5	4.2	16.6	18.4	21.5	24.4	27.0	36.7

Table V. NO_2 concentration (ppb) for Watertown, MA in non-gas cooking homes during the first week of winter sampling period.

				Percentiles					
	N	MEAN	SD	10	25	50	75	90	MAX
BEDROOM	75	8.2	3.5	4.5	5.2	7.1	10.8	13.5	17.0
ACT. ROOM	75	10.4	4.0	5.5	7.2	9.6	13.0	16.9	19.7
KITCHEN	75	10.6	4.4	5.5	6.7	9.9	13.0	16.4	21.7
OUTDOOR	74	20.6	5.1	14.3	16.8	20.5	24.9	26.4	31.0

Sixty-five percent of the outdoor values were within a 10 ppb range (15-25 ppb). The variation in indoor NO_2 levels was substantially greater in gas cooking homes than in electric homes.[2] For the gas cooking homes approximately 10% of the bedroom, 25% of the activity room and 50% of the kitchen concentrations exceeded the highest ambient concentration.

The week-to-week correlations among sampling locations were calculated to determine the variability of indoor concentrations over time. As shown in Tables VI and VII, the week-to-week correlations among outdoor samples and between indoor and outdoor samples were close to 0. None of these correlations was significant. On the other hand, the between-week comparison for the indoor samples showed high and significant ($p < .0001$) correlations. The stronger correlations were for the gas cooking homes. Indoor home samples within a given week had correlation coefficients greater than 0.76 for both home types.

These results raise the possibility that for the gas cooking homes, multiple location sampling and consecutive week monitoring are not providing substantially more information on indoor NO_2 concentrations beyond the one week sample in a single room. This issue will be explored further.[2] In St. Louis, Missouri and Kingston, Tennessee, a subset of homes have been monitored for eight to eighteen weeks over the course of a year, which will provide additional information on week-to-week variation.

Table VI. Correlations of winter week-long NO_2 levels by location for non-gas cooking homes. Paired samples N=72 to 79. All within home correlations significant at .0001 level.

WEEK 2	WEEK 1			
	OUTDOOR	BEDROOM	ACT. ROOM	KITCHEN
OUTDOOR	-.12	-.27	-.17	-.22
BEDROOM	.04	.68	.63	.54
ACT. ROOM	.11	.61	.79	.71
KITCHEN	.13	.57	.72	.73

Table VII. Correlations of winter week-long NO_2 levels by location for gas cooking homes. Paired samples N=173 to 184. All within home correlation significant at .0001 level.

WEEK 2	WEEK 1			
	OUTDOOR	BEDROOM	ACT. ROOM	KITCHEN
OUTDOOR	-.01	-.06	-.06	-.01
BEDROOM	-.04	.93	.79	.74
ACT. ROOM	-.03	.82	.93	.81
KITCHEN	-.03	.78	.82	.93

Particle concentrations were higher in homes with smokers than in homes without smokers by 30 to 35 ug/m^3 (Table VIII). Interestingly, the standard deviations in both home types were approximately 50% of the mean concentrations. The distribution was skewed for both smokers and non-smokers homes. This is expected since there were additional particle sources indoors, and even in the non-smokers home an occasional visitor may smoke. As shown in Table VIII the distribution of PM 2.5 concentrations was similar in weeks 1 and 2. About 50% of the low end of the distribution in homes with smokers overlapped the upper half of the concentrations in homes without smokers.

Table VIII. PM 2.5 concentrations (ug/m_3) in Watertown, MA homes during winter sampling period. NS = non-smoking and S = smoking homes.

HOME					PERCENTILES					
TYPE/WK		N	MEAN	SD	10	25	50	75	90	MAX
NS	WK 1	70	22.3	11.6	10.6	14.3	19.5	27.0	39.1	57.3
NS	WK 2	74	20.9	9.9	11.0	14.3	17.8	25.0	36.9	55.4
S	WK 1	147	53.3	25.1	25.2	35.5	49.9	66.9	91.9	144.7
S	WK 2	152	54.7	26.2	24.0	35.2	51.0	70.3	95.5	125.7

Intra-home variations in PM 2.5 concentrations could not be determined in this study because of the limited number of particle samplers. This issue will be examined in subsequent substudies. However, the week-to-week correlations were investigated. In the non-smoking homes the correlation was 0.45 and in the smoking homes the correlation was 0.71. These correlation coefficients were significantly different. This suggests a consistent influence of tobacco smoke in the Watertown winter PM 2.5 concentrations which, on average, more than doubled the indoor concentrations.

Curiously, two-way ANOVA of PM 2.5 by home classification categories indicated a small difference in PM 2.5 concentrations between gas and non-gas homes. For the first week sampled, gas homes had marginally significantly higher PM 2.5 levels than non-gas homes ($p = 0.06$ after controlling for smoking). The partial p-value for second week was 0.2. However, the t-test for difference in PM 2.5 between gas and non-gas homes was significant for both weeks, revealing a 10 ug/m^3 difference. At this point, we speculate that the higher PM 2.5 concentrations in gas homes resulted from heavier smoking among gas-cooking households. This will be examined in more detail.

REGRESSION ANALYSIS RESULTS

NO_2 Regression Analysis

A home characteristics questionnaire ascertained information on about 90 variables as noted below. In this analysis, only a preselected set of variables was completely verified and used in the regression analysis. The following were used as independent variables for NO_2 analysis: outdoor concentration; presence of stove and oven pilot lights where PILOT is an indicator variable 0 for no pilot lights or 1 for any pilot lights; number of hours of stove and oven use per week; the presence, type and use of kitchen ventilation fans; and house volume. These variables were regressed step-wise on the dependent variables of kitchen, activity room and bedroom NO_2 concentrations in separate linear regressions. The natural logarithmic transformed values of the dependent variables and the volume and hours of range use also were examined. One purpose of the regression modeling was to determine the predictive value of descriptor variables.

Table IX displays the bivariate correlation coefficients between the two weeks of NO_2 measurements and home characteristics variables for gas cooking homes. The presence of pilot light and hours of stove use did significantly influence NO_2 concentrations when examined by ANCOVA. Whereas, the presence and use of kitchen vent fan was not significant. However, the underlying relationship may have been missed because the distribution of pilot lights across kitchen vent fan use categories was unbalanced and PILOT may be serving as a surrogate variable for stove type.

Table IX. Correlation coefficients and P-value for NO_2 concentrations and descriptive variables: Number of pilot lights, number of hours of stove and oven use per week and house interior volume.

		NO_2 CONCENTRATIONS WEEK 1 AND 2					
		BD 1	BD 2	AR 1	AR 2	KT 1	KT 2
PILOT	r	.41	.41	.40	.38	.47	.46
	p	.0001	.0001	.0001	.0001	.0001	.0001
HR STV	r	.16	.15	.15	.16	.15	.12
	p	.02	.04	.05	.03	.04	.1
HR OV	r	.16	.18	.09	.12	.14	.14
	p	.03	.01	.2	.1	.06	.05
VOL	r	-.33	-.33	-.33	-.33	-.31	-.31
	p	.0001	.0001	.0001	.0001	.0001	.0001

In a two variable linear regression analysis restricted to gas homes only, pilot lights and house volume alone resulted in R-squares of 0.22, 0.24, 0.28 for bedroom, activity room and kitchen NO_2 concentrations. The addition of stove and oven use hours and outdoor NO_2 concentrations to the model only improved the R-square of the equation by 0.01 to 0.02. Table X summarizes the results of the NO_2 regression analysis by presenting the intercept, coefficients, R-square, root mean square error, mean of the dependent variable and coefficient of variance.

NO_2 concentrations in the three rooms were regressed separately for each of the two weeks. House volume is expressed as cubic feet of interior living volume not including basements or attics, unless the homeowner indicated that they were

used regularly. The HRS and HRO are the reported hours per week that the principle cook used the stove and oven. In this analysis, the pilot variable was reduced to a dichotomous variable of (0,1). As might be expected, the highest proportion of variance in NO_2 levels was accounted for in the models predicting kitchen values. The stability of the coefficients for the independent variables between weeks and among variables is encouraging. In subsequent physical models, inverse volume will be used along with measured air exchange rate. No attempt was made to specify the amount of gas consumed for cooking. Improved specificity of these variables may improve their predictive value.

Table X. Step-wise linear regression of home variables on indoor NO_2 concentrations for gas-cooking homes in winter sample, Watertown, MA.

N	LOCATION	WEEK	REGRESSION COEFFICIENTS INTERC	PILOT**	VOL**	HRSV*	HROV*	OUT*	R^2	DEP MEAN	RMSE	CV
152	BEDROOM	1	22.5	8.3	-.0006	.09	.19	.04	0.24	25.09	9.21	36.71
149	BEDROOM	2	16.9	8.6	-.0007	.12	.41	.3	0.26	25.63	9.77	38.12
150	ACT ROOM	1	24.8	10.1	-.0007	.09	.008	.12	0.25	28.07	10.36	36.89
151	ACT ROOM	2	21.8	9.2	-.0008	.18	.15	.25	0.24	28.54	10.73	37.59
151	KITCHEN	1	32.3	17.0	-.0009	.13	.22	.14	0.29	40.69	14.35	35.26
150	KITCHEN	2	34.6	16.7	-.001	.15	.37	.08	0.28	41.18	15.35	37.28

* $1 < P < 0.1$
** $P \leq 0.003$

Examination of the residual plots indicated a poor fit due to the skewed distribution of NO_2 concentrations and some independent variables. Thus, the regression analysis was performed using natural log transformed NO_2, volume and hours of oven and stove use. There was a substantial improvement in the overall R-squares of these models as compared to models with untransformed data. However, it is more difficult to interpret the coefficients of the derived non-linear models. Furthermore, the effect of independent variables on the dependent variables is no longer additive but multiplicative, which may be in contrast to the actual physical process. Table XI summarizes the results of the regression analysis in the gas homes, after logarithmic transformation of the variables.

Table XI. Multiple regression of log transformed NO_2 concentrations by location on home characteristics and log transformed outdoor NO_2 levels for Watertown, MA winter sampling. N = 150 to 153.

Dependent Variable	REGRESSION COEFFICIENTS					
	Intercept	Pilot* Light	ln (Volume)*	ln (NO_2 Outdoors)	ln (Hours of Stove Use)	R^2
ln (NO_2) Kitchen						
Week 1	5.70	0.48	-0.32	0.12 (p=0.37)	0.06 (p=0.18)	0.38
Week 2	6.11	0.46	-0.36	0.05 (p=0.76)	0.06 (p=0.16)	0.38
ln (NO_2) Act Room						
Week 1	4.99	0.39	-0.31	0.23 (p=0.09)	0.06 (p=0.20)	0.32
Week 2	5.23	0.34	-0.35	0.25 (p=0.07)	0.09 (p=0.04)	0.33
ln (NO_2) Bedroom						
Week 1	5.30	0.38	-0.33	0.11 (p=0.39)	0.08 (p=0.05)	0.32
Week 2	4.98	0.39	-0.35	0.29 (p=0.04)	0.08 (p=0.05)	0.36

* $p \leq 0.001$

The models for different locations and for different weeks showed stable coefficients. It is not clear, however, why the variable "hours of stove use" did not show significant contribution on the NO_2 kitchen levels, while it did have a significant effect on the NO_2 levels in the other locations.

A substantially larger proportion of the variance could be explained when data for the non-gas homes were included in the regression analysis, setting "number of pilot lights" and "hours of stove/oven use" equal to zero for the non-gas houses. Thus, the variable "hours of stove use" represents a surrogate for NO_2 production due to cooking. An example of such a model is:

$$\ln (NO_2)\ \text{kitchen} = 1.29 + 0.64\ \text{pilot}^* + 0.36 \ln (NO_2\ \text{outdoors})^* + 0.21 \ln (\text{hours of stove use})^* + 0.12 \ln (\text{hours of oven use})^{**}$$

$R^2 = 0.73$ * $p \leq 0.001$, N = 216 ** p = 0.04

Similar models were obtained for the other locations and weeks, with R-square ranging from 0.63 to 0.74.

PM 2.5 Regression Analysis

As a preliminary screening, bivariate correlation coefficients were calculated between the PM 2.5 data and the home factor variables i.e., pilot light, fan use, hours of stove and oven use, volume of the home and number of cigarettes smoked in the home per week. Cigarettes/week and house volume were strongly correlated with PM 2.5 concentrations. The weak correlations with the other variables seemed spurious since they disappeared in subsequent multiple regression.

Linear and log transformed multiple regression analysis to develop predictive models for indoor PM 2.5 levels was carried out. The linear regression model restricted to just weekly cigarette consumption and house volume was run for each week with the following results:

Week 1 $PM\ 2.5 = 43.4 + 0.11\ (Cig/W) - 1.4\ (Vol/1000\ ft^3)$
N = 183 $R^2 = 0.34$ RMSE = 20.4

Week 2 $PM\ 2.5 = 44.9 + 0.12\ (Cig/W) - 1.3\ (Vol/1000\ ft^3)$
N = 191 $R^2 = 0.32$ RMSE = 21.9

The contribution of cigarettes to the indoor PM 2.5 concentrations converted to a more conventional unit of cigarettes consumed per day indicates about 0.77 ug/m^3 per cigarette smoked per day. Thus, smoking a pack-a-day at home would increase the indoor concentrations 15.5 ug/m^3 on average, according to these models. Again, logarithmic transformations were performed on the data to obtain a better fit for the models. The following models were obtained for Week 1 and Week 2:

Week 1 $\ln (PM\ 2.5) = 5.95 - 0.35 \ln (Volume)^* + 0.19 \ln (Cig/week)^* + 0.01\ PM\ 2.5\ outdoors^{**}$
N = 186 $R^2 = 0.47$ * $p \leq 0.001$ ** p = 0.09

Week 2 $\ln (PM\ 2.5) = 5.83 - 0.36 \ln (Volume)^* + 0.20 \ln (Cig/week)^* + 0.03\ PM\ 2.5\ outdoors^*$
N = 194 $R^2 = 0.55$ * $p \leq 0.001$

RESULTS OF ACUTE HEALTH SURVEY

Respiratory health data were obtained for 309 children over a period of 151 days from December 1, 1984 to April 30, 1985, resulting in a total number of observations of 44,654 children days. This paper presents the results for the 266 children who participated in the Acute Health Survey as well as in the Indoor Air Monitoring Sampling. Symptoms B and C (of Table II) were classified as symptoms of upper respiratory illness (URS) and symptoms D, E and F as lower respiratory illness (LRS). This scheme for classification of symptoms into upper and lower respiratory diseases was proposed by Monto et al, 1971 and later used by Tager and Speizer, 1976 in a study of respiratory illness in East Boston.[18,19] A severity score for URS and LRS was determined, based on diary entries L, N and P (Table I), resulting in four levels of severity. A "0" indicates the absence of respiratory symptoms. A "1" indicates symptoms but without additional health care (L, N or P entry). If the symptom restricted activities (L), a severity of "2" was assigned. If the symptoms required a visit to the doctors (N) a severity level "3" was given. Severity "4" was assigned when symptoms resulted in hospitalization (P). Furthermore, the number of events of upper and lower respiratory illness was determined, using the following concept of disease event. When respiratory symptoms were reported on a day, while on the two previous days the child was reported to be free of symptoms, this was considered to be a new event of respiratory illness (URE and LRE for events of upper respectively lower respiratory illness).

Table XII shows the frequency distribution of URS and LRS over days of observation by severity score.

Table XII. Frequency distributions (%) of reported upper (URS) and lower (LRS) respiratory symptoms by severity score between December 1, 1984 and April 30, 1985 for 266 children in Watertown.

SEVERITY	URS	LRS	DESCRIPTOR
0	91.37	98.45	NO ILLNESS
1	7.49	1.35	SYMPTOM WITHOUT ADDITIONAL HEALTH CARE
2	0.90	0.16	RESTRICTED ACTIVITIES
3	0.22	0.05	DOCTOR VISIT
4	0.02	0.003	HOSPITALIZATION

Of the 266 children participating in both the health and indoor air pollution survey, 234 (88%) reported at least one day of upper respiratory symptoms during this 151 day winter sampling period. On the other hand, only 58 (22%) reported at least one day of lower respiratory symptoms. The median frequency of separate upper respiratory events was three accounting for a median of eight days of symptoms. Eighty-eight percent of the children had fewer than two lower respiratory events with the majority (78%) reporting no events.

These distributions are clearly skewed. Prior to an analysis of the potential relationship between the acute health effects and exposure to indoor pollutants, the calendar data will be further examined along with the questionnaire data and pulmonary function results from the first and second annual exams. This effort will provide a better perspective on the meaning of acute reversible health data in relation to indicators of chronic respiratory conditions. In addition, such an effort may validate the data and indicate situations of over- or under-reporting, and/or chronic conditions unrelated to air pollution ex-

posure. Furthermore, the calendar data will be analyzed in relation to daily meteorological data, i.e., temperature, humidity, wind speed, etc., and daily ambient air pollution data.

DISCUSSION

The purpose of this paper is to describe Harvard's Indoor Air Quality Acute Health Study. The analysis of the winter survey conducted in Watertown, Massachusetts is presented for illustrative purposes only. Since the study design is based on one complete year's worth of health and pollution data collected on a targeted sample of 300 in each of six U.S. cities studied, we do not draw inferences based on the relationships presented in this paper. Furthermore, the week-to-week correlations for NO_2 and PM 2.5 suggest the need to reconsider the consecutive week sampling protocol. This issue will be addressed in special substudies of over 100 homes that will have at least eight weeks of monitoring over a year.

Other important health and air pollution issues need to be addressed. The health survey relies on self-administered diary data. To date, the magnitude of the study has not permitted time to develop a scheme for verifying health responses by direct and periodic visits to the child's home. Room-to-room variations in NO_2 clearly exist, however, we currently do not have the sampling equipment to measure the within-home variation in PM 2.5 concentrations. A substudy of multiple room sampling is being planned. It is our intention to develop physical models for predicting indoor NO_2 and PM 2.5 concentrations once the air exchange information is available. The ambient, school, and home concentration data will be utilized to develop a microenvironmental exposure model for each child. A possible modeling construct has already been described in a paper by Letz et al 1985.[20] Indoor particle samples will be analyzed for elemental composition using XRF techniques and for acidity (pH) when collected using a denuder pre-sampler. Analysis of these data will offer insight on the penetration of ambient particulate matter and the qualitative contribution of sources.

Finally, as we have continued the study in Kingston, Tennessee we have altered the design to take advantage of the high prevalence of wood stoves and kerosene heaters. The sample in this city was stratified to provide a comparison among these sources as well as cigarette smoking. Working with researchers at Oak Ridge National Labs the Tennessee survey was expanded to include radon, formaldehyde, polynuclear aeromatic compounds, and sampling for biological agents such as molds, bacteria and allergens.

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